# Electrocardiographic Findings in Patients with Complete Atrioventricular Block*

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The introduction of the artificial electrical pacemaker for the treatment of patients with complete A-V block has been accompanied by renewed interest in the pathogenesis of this disorder. It has been considered that most cases of complete block are due to organic or functional lesions either in the A-V node or in the common A-V bundle. Barker and Hirschfelder in 1909 and later Eppinger and Rothberger (1910) demonstrated experimentally that section of both bundle-branches or of the bundle of His could produce a complete A-V block. Wilson and Herrmann in 1921 studied this problem again and concluded that complete block could be due to an interruption of both bundle-branches. They also postulated that in complete block, if the ventricular complexes were of an aberrant type, bilateral bundle-branch block must exist, whereas if the ventricular complexes were of normal contour the interruption must have been above the bifurcation. Yater, Cornell, and Claytor in 1936 published a review of the published reports of 48 patients with pathologically proven complete A-V block due to bilateral bundle-branch block. On the basis of their studies, they concluded that a large number of patients with complete A-V block would have an interruption of both bundle-branches rather than a block above the bifurcation.

Rosenbaum and Lepeschkin in 1955 described two cases of bilateral bundle-branch block. By careful analysis of the electrocardiographic tracings, they formulated criteria for the diagnosis of bilateral bundle-branch block. They stated that this diagnosis could be made when one branch was completely interrupted and the other incompletely or intermittently interrupted. Further, bilateral bundle-branch block could be diagnosed when the

conduction through both bundle-branches was unequally impaired. In both of these situations, one would observe a complete right or left bundle-branch block with prolongation of the P-R interval. Complete block would be the manifestation of failure of conduction through the more depressed branch, while prolongation of the P-R interval would be the manifestation of depressed conduction through the less impaired branch. They stressed that true bilateral bundle-branch block must show a prolonged A-V conduction time.

Mahaim (1931), and more recently Lenègre and Moreau (1962), and Lenègre (1964), have conducted pathological studies in the hearts of patients who had complete A-V block. They confirmed the electrocardiographic impression that in the great majority of patients this type of block was due to an anatomical bilateral bundle-branch block.

To elucidate further the relation between bilateral bundle-branch block and A-V block, we have analysed the records of all patients with complete A-V block admitted to the University Hospital, Saskatoon, in the five-year period between 1960 and 1965. Patients with surgical A-V block and those in whom the history and clinical diagnosis suggested digitalis toxicity were excluded. The results of our study form the basis of this communication.

### SUBJECTS AND RESULTS

Between 1960 and 1965, 57 patients with an electrocardiographic diagnosis of complete A-V block were studied at the University Hospital. In many instances previous electrocardiograms were available. The age of the patient, the clinical history, and electrical axis in the frontal plane were carefully recorded.

The 57 patients were divided into two main groups, according to the contour of the ventricular complexes during the periods of complete A-V

TABLE

CLINICAL AND ELECTROCARDIOGRAPHIC DATA OF 57 PATIENTS WITH COMPLETE A-V BLOCK

| Group | Sub-group | No. of patients | QRS contour during A-V block | QRS contour of conducted beats | Axis in frontal plane of con-ducted beats | Average age (yr.) | Adams–Stokes attacks | % of total series |
|---|---|---|---|---|---|---|---|---|
| A | 1 | 24 | Aberrant | Right bundle-branch block | −15°; −110° | 74 | Yes | 42 |
|   | 2 | 7 | Aberrant | Left bundle-branch block | −25°; −80° | 74 | Yes | 12 |
|   | 3 | 14 | Aberrant | — | — | 74 | Yes | 25 |
| B | 1 | 5 | Normal | Normal | −15°; −30° | 73 | Yes | 9 |
|   | 2 | 7 | Normal | Normal | +30°; +80° | 25 | No | 12 |

block (Table). Group A consisted of 45 patients in whom the ventricular complexes had an aberrant contour and a prolonged duration (more than 0·12 sec.). Group B consisted of 12 patients with ventricular complexes of normal contour and duration (less than 0·12 sec.).

*Group A.* Clinically, the 45 patients in this group were remarkably similar. Their ages at the time of admission varied between 41 and 87 years, with an average of 74 years. Only one patient has been excluded in calculating the average age; this was a 6-year-old boy who will be discussed later. All of these patients had severe Adams–Stokes attacks. The mean electrical axis of the QRS in the frontal plane varied between − 15 and − 110 degrees. Of the 45 patients in this group, 31 had been seen in this hospital before, and electrocardiograms obtained on previous occasions showed a normal 1:1 A-V conduction in the presence of a

bundle-branch block; 24 had a right bundle-branch block (Group A, subgroup 1) and 7 patients had a left bundle-branch block (Group A, subgroup 2).

The pattern of bundle-branch block (right or left) was the only abnormality observed before the development of complete A-V block in 9 patients. However, 18 patients showed an evolution through first- and/or second-degree A-V block (Fig. 1 and 2). One of these patients showed alternating periods of normal and aberrant ventricular complexes of the conducted beats (Fig. 3). The remaining 4 patients in these two subgroups presented from the beginning with electrocardiographic changes of intermittent or incomplete A-V block. The contour of the ventricular complexes during periods of complete A-V block was always aberrant and differed from the contour of the conducted beats.
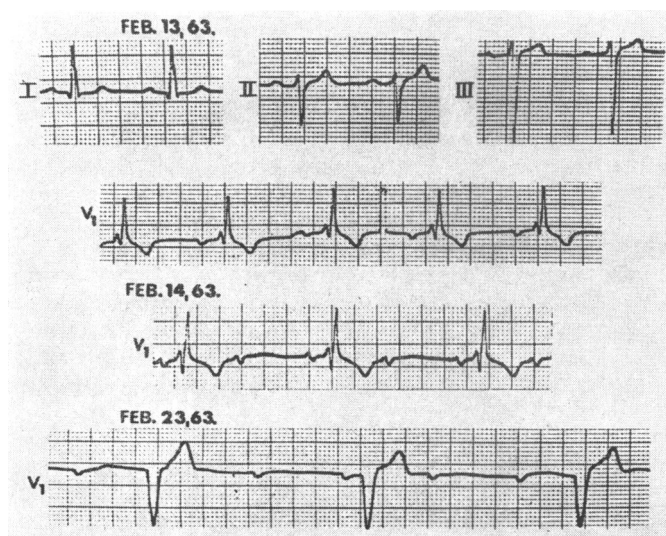
The 14 patients of Group A, subgroup 3, pre-



FIG. 1.—Group A, subgroup 1. Right bundle-branch block with left axis deviation and normal P–R interval. Next day, right bundle-branch block with 2:1 A-V block, and later complete A-V block. Patient developed Stokes–Adams attacks and needed pacing.
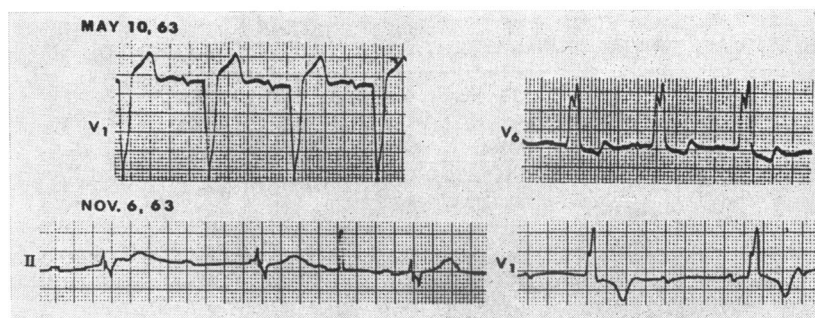
FIG. 2.—Group A, subgroup 2. Left bundle-branch block and first-degree A-V block. Six months later complete A-V block. Patient developed Adams–Stokes attacks and needed pacing.
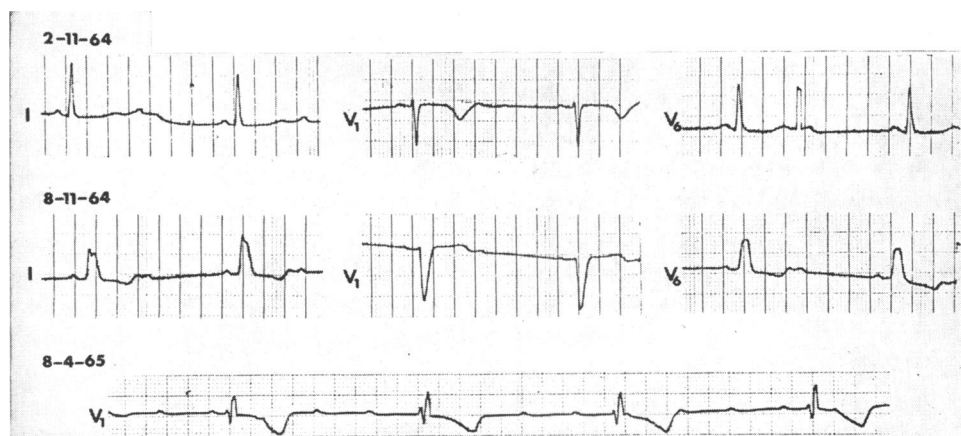


FIG. 3.—Group A, subgroup 2. 2:1 A-V block with normal contour of ventricular complexes. Six days later 2:1 A-V block and left bundle-branch block. Five months later complete A-V block. Cardiac pacemaker had to be implanted.

sented with electrocardiographic findings of complete A-V block and no previous records were available for comparison and analysis.

In only 3 patients of this group was a myocardial infarction considered to be the cause of the block (Fig. 4). In all these patients the necropsy showed extensive antero-septal infarction, with destruction of both bundle-branches. Two patients died during the acute stage of infarction and the third died three years later.

*Group B.* This group included 12 patients with normal contour and duration of the ventricular beats during periods of complete A-V block. The group was subdivided into two subgroups: subgroup 1 consisted of 5 patients (ages 68 to 81 years) who had had severe Adams–Stokes attacks. Three patients had had second-degree A-V block for several months and their clinical and electrocardiographic findings

did not suggest the presence of myocardial infarction. The mean electrical axis in the frontal plane was between $-15$ and $-30$ degrees (Fig. 5). The remaining two patients had complete A-V block and evidence of recent posterior wall myocardial infarction. After four to five days in hospital the complete A-V block changed to second-degree block (with Wenckebach periods), then to first-degree block, and finally to normal 1:1 A-V conduction (Fig. 6). Subgroup 2 consisted of 7 patients who were younger at the time of their first admission. Their ages varied between 6 and 50 years (average 25). Their mean electrical axis in the frontal plane varied between $+30$ and $+80$ degrees. Two of these patients had had, for years, alternating periods of intermittent and complete A-V block, and the other 5 had always shown complete A-V block in the electrocardiogram (Fig. 7). They all had a history of a slow pulse for many years, and
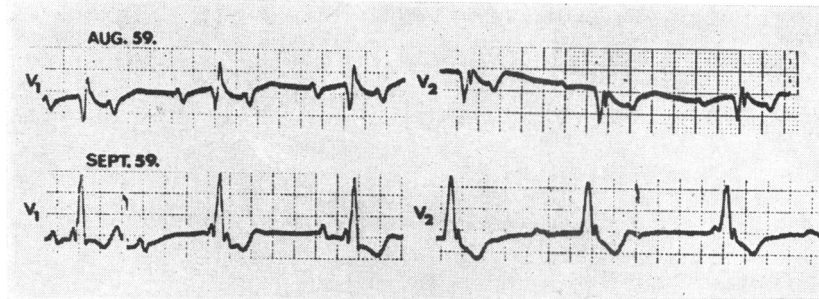
FIG. 4.—Group A. Aug. 1959: antero-septal myocardial infarction with right bundle-branch block and 2:1 A-V block. Sept. 1959: complete A-V block with Adams–Stokes attacks.

in 3 of them associated cardiac anomalies had been found at cardiac catheterization (corrected transposition of the great vessels in 2 patients and inter-atrial septal defect in 1). None of these patients suffered from fainting spells. The electrocardiographic findings and the slow pulse had been discovered when they sought medical attention for unrelated complaints. Clinically, they have all been diagnosed as congenital A-V block.

## DISCUSSION

It is known that the cells of the bundle of His possess a higher degree of automatism than do those cells situated in either bundle-branch. It is reasonable to believe that when the interruption of the conducting system is situated above the bifurcation, then the ventricles will be driven by a pacemaker which is also situated above the bifurcation. Thus the ventricular complexes should be of normal con-

tour. On the other hand, if the interruption of the conducting system is below the bifurcation, then the ventricles must be driven by a pacemaker which is situated either in a bundle-branch or in some of the Purkinje cells in the ventricular walls. Thus the ventricular complexes should be of an aberrant type (Wilson and Herrmann, 1921).

The electrocardiographic changes in the 31 patients of Group A (subgroups 1 and 2) seem to have gone through at least three stages (Fig. 8). *The first stage* was characterized by the presence of either right or left bundle-branch block with normal conduction through the opposite intact bundle. In *the second stage*, the bundle-branch block had persisted but there was a conduction delay through the opposite bundle-branch. The latter was manifested in the electrocardiogram by the presence of a first- and/or second-degree A-V block. In two-thirds of the patients in whom this second stage
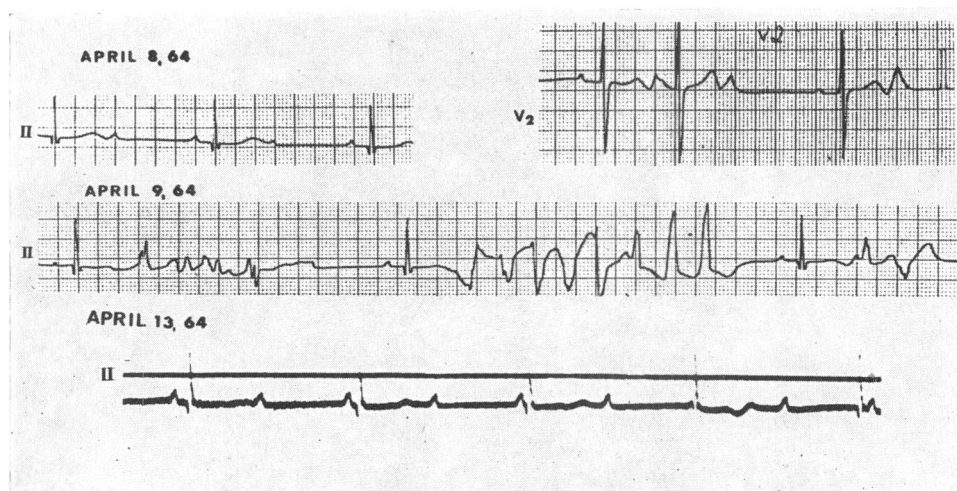
FIG. 5.—Group B, subgroup 1. Second-degree A-V block with 2:1 and 3:2 A-V conduction. Next day ectopic ventricular beats with runs of ventricular tachycardia. Later complete A-V block. A pacemaker was implanted.
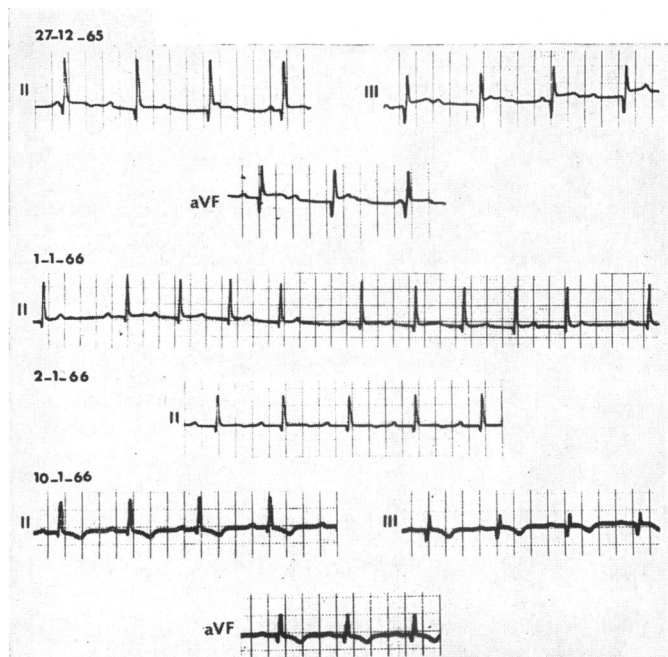
FIG. 6.—Group B, subgroup 1.  A-V dissociation due to a combination of A-V block and acceleration of an A-V junctional pacemaker.  Recent posterior wall myocardial infarction.  Later second-degree A-V block of the Wenckebach type and next day first-degree A-V block.  Later normal A-V conduction.  Patient recovered.
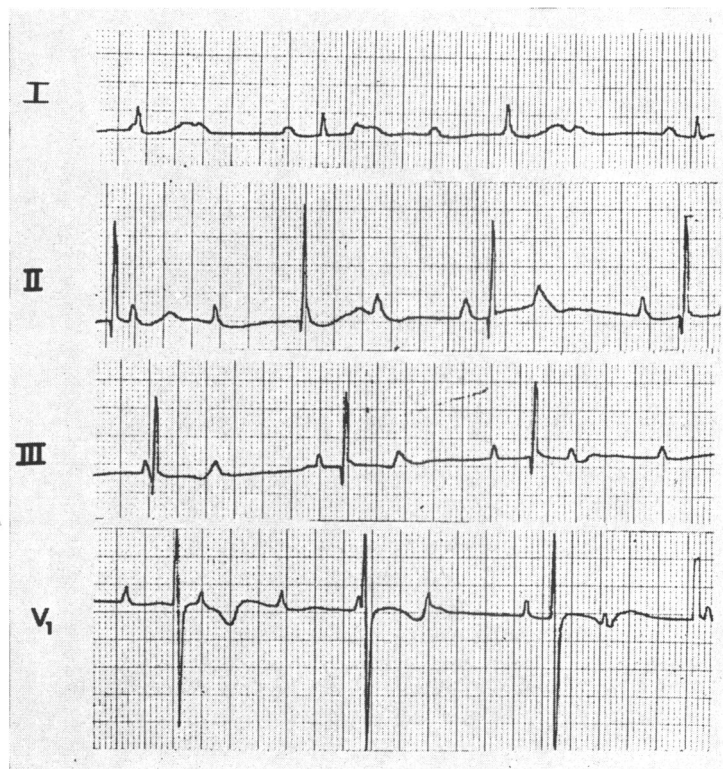


FIG. 7.—Group B, subgroup 2.  Sinus arrhythmia with complete A-V block and intermediate electrical axis. Congenital A-V block.
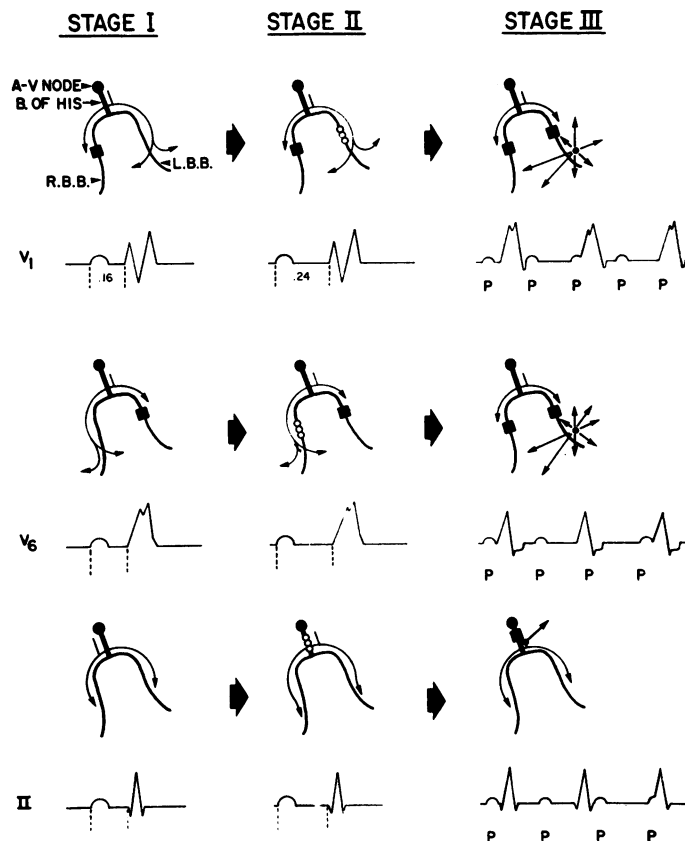
FIG. 8.—Diagrammatic representation of the three stages of the electrocardiogram in patients with complete (third-degree) A-V block. See text for further discussion.

was documented, a 2:1 A-V block was present. In the remaining one-third, a Mobitz type II A-V block was present. In no patient in this series was a Mobitz type I block (with Wenkebach periods) found. This is in agreement with previous reports that type I A-V block is rare in cases of Adams–Stokes disease and that type II A-V block is commonly found before complete A-V block (Gilchrist, 1958; Kaufman *et al.*, 1961; Donoso, Adler, and Friedberg, 1964; Langendorf and Pick, 1964). In *the third stage*, both bundle-branches were blocked and the ventricles were driven by a subsidiary ventricular pacemaker. The fact that the ventricular complexes during the third stage had a different contour from that seen during the first and second stages speaks against the possibility of unilateral bundle-branch block and complete A-V block with the pacemaker above the bifurcation.

Nine patients in this group had a bundle-branch block which was not associated with an abnormality of the A-V conduction before the complete A-V

block appeared. It is possible that they had had a bundle-branch block and that a block of the opposite bundle had occurred shortly thereafter (precipitating complete A-V block), or that the block on the opposite side was only intermittently present and that the records were obtained during periods of normal A-V conduction. In this series, right bundle-branch block was the most common antecedent of bilateral bundle-branch block. Periods of complete A-V block were intermittently present early in the course of the disease, and they alternated with periods of first- and/or second-degree A-V block. This suggests that while the first stage of disease was permanent, there was a period of instability during the second and third stages. The second stage is not always characterized by the presence of a permanent block in one of the bundles and a partial one in the other. As several investigators have demonstrated (Strauss and Langendorf, 1943; Rosenbaum and Lepeschkin, 1955; Unger *et al.*, 1958; Lepeschkin, 1964), the block could be
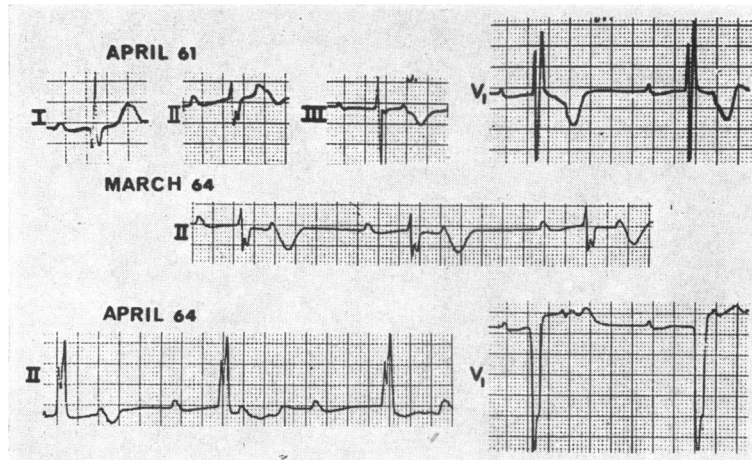
FIG. 9.—Right bundle-branch block with left axis deviation and 2:1 A-V block. In 1964 complete A-V block. A cardiac pacemaker had to be inserted.

partial in both bundles (more in one than in the other) and the patient might remain indefinitely in the second stage of disease. In these cases the electrocardiogram would show a bundle-branch block with an A-V conduction delay or alternating periods of right and left bundle-branch block. The clinical aspects of this group coincide with those reported by other authors (Campbell, 1944; Gilchrist, 1958; Rowe and White, 1958).

The 14 patients of Group A, subgroup 3, were found to have complete A-V block, but no previous records were available. The ventricles were driven by a pacemaker which seemed to be below the bifurcation. It is very probable that at least some of these patients had bilateral bundle-branch block which was not documented at an earlier stage. Some of these patients might have had a block above the bifurcation with the ventricles driven by a focus in the ventricular wall. The similarity of these 14 patients to the other 31 of Group A suggests that they also had bilateral bundle-branch block. With this assumption, 45 patients (78%) had in effect bilateral bundle-branch block (Table).

In all patients of Group A, the mean electrical axis in the frontal plane was deviated to the left, though it is possible that this was a manifestation of some degree of left ventricular hypertrophy in patients in the seventh or eighth decade of life; the same finding was also present in a child of 8 years of age (Fig. 9) who had no other evidence of left ventricular hypertrophy. Recently, Wigle and Baron (1966) found that surgical incision of the anterior division of the left bundle-branch resulted in left axis deviation in the electrocardiogram. Perhaps the patients in this series had a

block involving the anterior division of the left bundle-branch during the first stage of the disease.

Ventricular complexes of normal duration and contour in the presence of complete A-V block suggest that the origin of the subsidiary ventricular pacemaker is above the bifurcation. Wilson and Herrmann (1921) thought that occasionally ventricular complexes of normal contour and duration could be seen in experimental sections of both bundle-branches. They postulated that two pacemakers, one on each bundle acting synchronously, could result in a ventricular complex of normal duration. However, two pacemakers acting simultaneously is a very remote possibility and this could only occur by pure chance. Yater et al. (1936) explained this possibility by suggesting the presence of one pacemaker in one of the bundle-branches sending impulses directly to the Purkinje network of the other side through the interventricular septum. One last possibility is the presence of a focus situated in the septum, below the bilateral bundle-branch block, in a position equidistant from both bundle-branches. In this study 12 patients had QRS complexes of normal contour and duration. The 5 patients in Group B, subgroup 1, had had conducted beats which also showed normal contour and duration. For this reason the possibility of bilateral bundle-branch block had to be excluded. These 5 patients behaved clinically in a similar way to those patients described previously with bilateral bundle-branch block. While there is no histological evidence, these 5 patients (9% of the total series) do appear to have suffered from acquired heart disease with the block above the bifurcation (Fig. 5).

Myocardial infarction was a relatively uncommon cause of A-V block in this series. As Blondeau, Rizzon, and Lenègre (1961) have reported, patients with posterior wall myocardial infarction suffer from a partial and transitory impairment of the conduction system, generally above the bifurcation (Group B). In patients with antero-septal myocardial infarction, the destruction of the conduction system is more extensive and involves both bundle-branches (Group A).

As other authors have found, congenital A-V block is almost always due to a complete or incomplete developmental defect of the common A-V bundle (Langendorf and Pick, 1954; Campbell and Thorne, 1956; Donoso *et al.*, 1956; Paul, Rudolf, and Nadas, 1958; Smithells and Outon, 1959; Nakamura and Nadas, 1964). Patients with congenital A-V block do not have serious Adams–Stokes attacks. It is possible that some of these patients may have Adams–Stokes attacks in later life when the cells of the common A-V bundle have lost some automaticity. However, no patient in our series suggested this possibility.

One patient in Group A deserves special comment. This is a 6-year-old boy who suffered from Adams–Stokes attacks and who required the implantation of an internal pacemaker (Fig. 9). The young age suggests that he had a congenital A-V block; however, analysis of his previous records revealed that for several years he had right bundle-branch block with left axis deviation and a second-degree A-V block (second stage of bilateral bundle-branch block). All criteria, apart from age, suggested that this patient had the acquired type of bilateral bundle-branch block. The findings and the clinical behaviour of this unusual patient suggest to us that he might have suffered from a type of cardiomyopathy involving both bundle-branches.

Another patient who seemed to be unique in this series also belonged to Group A (Fig. 3). This patient had a second-degree A-V block with ventricular complexes whose contour alternated between normal and aberrant. These changes were present for approximately six months before she developed complete A-V block. It seems that this patient had two separate areas of partial block, one above the bifurcation and the other in the left bundle-branch. The block above the bifurcation seems to have been more permanent and was clearly manifested when the left bundle-branch was conducting normally. The cause of the complete A-V block may have been a complete block in the common A-V bundle or the development of a block in the right bundle-branch, leading to bilateral bundle-branch block.

## SUMMARY AND CONCLUSIONS

The review and analysis of the electrocardiographic tracings of 57 patients with complete A-V block studied over a five-year period at the University Hospital, Saskatoon, indicate that 45 patients (79% of the total) had a bilateral bundle-branch block. Patients with bilateral bundle-branch block seem to go through three stages. The first stage is characterized by the presence of a bundle-branch block; in the second stage the bundle-branch block is complicated by a first- and/or second-degree A-V block; and in the third stage both bundle-branches are blocked and a subsidiary ventricular pacemaker (with aberrant ventricular complexes) drives the ventricles. Five patients (9% of the total) had an acquired A-V block situated above the bifurcation. In only 5 patients was myocardial infarction considered to be the cause of the A-V block. In the 7 patients (12% of the total) with congenital A-V block, the interruption of the conduction system was situated above the bifurcation. Clinically, these patients were quite distinct from the other groups.

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